	Type	L #	Hits	Search Text	DBs	Time Stamp	Comments	Error Definition	Errors
1	BRS	L1	583	calcineurin	USPAT; US-PGPUB; EPO; JPO; DERWENT	2002/10/18 17:30			0
2	BRS	L2	0	calcipressin	USPAT; US-PGPUB; EPO; JPO; DERWENT	2002/10/18 17:31			0
3	BRS	L3	0	1 same (endogenous adj inhibitor)	USPAT; US-PGPUB; EPO; JPO; DERWENT	2002/10/18 17:32			0
4	BRS	L4	0	csp adj 1	USPAT; US-PGPUB; EPO; JPO; DERWENT	2002/10/18 17:32			0

> d his

(FILE 'HOME' ENTERED AT 17:35:59 ON 18 OCT 2002)

FILE 'MEDLINE, CAPLUS, BIOSIS, EMBASE, SCISEARCH, AGRICOLA' ENTERED AT
17:36:27 ON 18 OCT 2002
L1 13784 S CALCINEURIN
L2 26 S L1 (P) (ENDOGENOUS INHIBITOR)
L3 6 DUPLICATE REMOVE L2 (20 DUPLICATES REMOVED)
L4 18 S CALCIPRESSIN
L5 144 S CSP1
L6 0 S L5 (P) L1
L7 16 S L4 (P) L1
L8 4 DUPLICATE REMOVE L7 (12 DUPLICATES REMOVED)
L9 4 S L8 NOT L3

=> log y

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FILE 'HOME' ENTERED AT 17:35:59 ON 18 OCT 2002

=> file medline caplus biosis embase scisearch agricola
COST IN U.S. DOLLARS                                SINCE FILE      TOTAL
                                                       ENTRY    SESSION
FULL ESTIMATED COST                                     0.21       0.21

FILE 'MEDLINE' ENTERED AT 17:36:27 ON 18 OCT 2002

FILE 'CAPLUS' ENTERED AT 17:36:27 ON 18 OCT 2002
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FILE 'AGRICOLA' ENTERED AT 17:36:27 ON 18 OCT 2002

=> s calcineurin
L1        13784 CALCINEURIN

=> s l1 (p) (endogenous inhibitor)
L2           26 L1 (P) (ENDOGENOUS INHIBITOR)

=> duplicate remove l2
DUPLICATE PREFERENCE IS 'MEDLINE, CAPLUS, BIOSIS, EMBASE, SCISEARCH'
KEEP DUPLICATES FROM MORE THAN ONE FILE?  Y/(N):n
PROCESSING COMPLETED FOR L2
L3             6 DUPLICATE REMOVE L2 (20 DUPLICATES REMOVED)

=> d l3 1-6 ibib abs

L3   ANSWER 1 OF 6     MEDLINE                              DUPLICATE 1
ACCESSION NUMBER:    2002183217     MEDLINE
DOCUMENT NUMBER:     21895837   PubMed ID: 11786544
TITLE:               Targeted inhibition of calcineurin in pressure-overload
                     cardiac hypertrophy. Preservation of systolic function.
AUTHOR:              Hill Joseph A; Rothermel Beverly; Yoo Ki-Dong; Cabuay
                     Barry; Demetroulis Elaine; Weiss Robert M; Kutschke
                     William; Bassel-Duby Rhonda; Williams R Sanders
CORPORATE SOURCE:    Department of Internal Medicine, University of Iowa College
                     of Medicine, Iowa City, Iowa 52242-1081, USA..
                     joseph-hill@uiowa.edu
CONTRACT NUMBER:     HL03908 (NHLBI)
SOURCE:              JOURNAL OF BIOLOGICAL CHEMISTRY, (2002 Mar 22) 277 (12)
                     10251-5.
                     Journal code: 2985121R. ISSN: 0021-9258.
PUB. COUNTRY:        United States
DOCUMENT TYPE:       Journal; Article; (JOURNAL ARTICLE)
LANGUAGE:            English
FILE SEGMENT:        Priority Journals
ENTRY MONTH:         200204
ENTRY ...

... of myocyte transformation. It is not known whether overexpression of
MCIP1, a recently described   ***endogenous***     ***inhibitor***   of
  ***calcineurin***  , impacts the hypertrophic response to
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of hemodynamic stress are unknown. Transgenic mice expressing a human cDNA encoding hMCIP1 in the myocardium were subjected to thoracic aortic banding. Transgenic mice and wild type littermates tolerated pressure overload equally well. Wild type mice developed left ventricular hypertrophy, but the hypertrophic response in transgenics was significantly blunted. An isoform of MCIP1 transcript was up-regulated by pressure stress, whereas MCIP2 transcript was not. Expression patterns of fetal genes were differentially regulated in banded MCIP1 hearts compared with wild type. Echocardiography performed at 3 weeks and 3 months revealed preservation of both left ventricular size and systolic function in banded MCIP1 mice despite the attenuated hypertrophic response. These data demonstrate attenuation of hypertrophic transformation when ***calcineurin*** is inhibited by MCIP1. Further, these data suggest that activation of hypertrophic marker genes may not be directly dependent on ***calcineurin*** activity. Finally, they demonstrate that ventricular performance is preserved despite attenuation of compensatory hypertrophy.

L3 ANSWER 2 OF 6 MEDLINE DUPLICATE 2
ACCESSION NUMBER: 2002397693 MEDLINE
DOCUMENT NUMBER: 22133761 PubMed ID: 12114545
TITLE: Calpain-dependent cleavage of cain/cabin1 activates calcineurin to mediate calcium-triggered cell death.
AUTHOR: Kim Min-Jung; Jo Dong-Gyu; Hong Gil-Sun; Kim Byung Ju; Lai Michael; Cho Dong-Hyung; Kim Ki-Woo; Bandyopadhyay Arun; Hong Yeon-Mi; Kim Do Han; Cho Chunghee; Liu Jun O; Snyder Solomon H; Jung Yong-Keun
CORPORATE SOURCE: Department of Life Science, Kwangju Institute of Science and Technology, 1 Oryong-dong, Puk-gu, Kwangju 500-712, Korea.
SOURCE: PROCEEDINGS OF THE NATIONAL ACADEMY OF SCIENCES OF THE UNITED STATES OF AMERICA, (2002 Jul 23) 99 (15) 9870-5. Journal code: 7505876. ISSN: 0027-8424.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200209
ENTRY DATE: Entered STN: 20020731
Last Updated on STN: 20020906
Entered Medline: 20020904

AB Cain/cabin1 is an ***endogenous*** ***inhibitor*** of ***calcineurin*** (Cn), a calcium-dependent serine/threonine phosphatase involved in various cellular functions including apoptosis. We show here that during apoptosis cain/cabin1 is cleaved by calpain at the carboxyl terminus to generate a cleavage product with a molecular mass of 32 kDa as a necessary step leading to Cn-mediated cell death. Mouse cain/cabin1 was identified from a thymus cDNA library by an in vitro substrate-screening assay with calpain. Exposure of Jurkat cells to the calcium ionophore, induced cain/cabin1 cleavage and cell death, accompanied by activation of calpain and Cn. The calpain inhibitors, calpeptin and ZLLY, suppressed both -induced cain/cabin1 cleavage and Cn activation, indicating that Cn activation and cain/cabin1 cleavage are calpain-dependent. Expression of cain/cabin1 or a catalytically inactive Cn mutant [CnA beta(2)(1-401/H160N)] and treatment with FK506 reduced -induced cell death. In vitro calpain cleavage and immunoprecipitation assays with deletion mutants of cain/cabin1 showed that cleavage occurred in the Cn-binding domain of cain/cabin1, indicating that the cleavage at its C terminus by calpain prevented cain/cabin1 from binding to Cn. In addition, in vitro binding assays showed that cain/cabin1 bound to the Cn B-binding domain of Cn A. Taken together, these results indicate that calpain cleaves the ***calcineurin*** -binding domain of cain/cabin1 to

[redacted]
DOCUMENT NUMBER: [redacted] PubMed ID: [redacted]
TITLE: Thapsigargin induced apoptosis involves Cabin1-MEF2-mediated induction of Nur77.
AUTHOR: Liu W; Youn H D; Liu J O
[redacted]

02139, USA.
SOURCE: EUROPEAN JOURNAL OF IMMUNOLOGY, (2001 Jun) [redacted](6) 1757-64.
Journal code: 1273201. ISSN: 0014-2980.
PUB. COUNTRY: Germany: Germany, Federal Republic of
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200108
ENTRY DATE: Entered STN: 20010813
Last Updated on STN: 20010813
Entered Medline: 20010809

AB Thapsigargin (TG), which inhibits endoplasmic reticulum-dependent Ca(2 +)-ATPase and thereby increases cytosolic Ca(2 +), has been reported to cause apoptosis in T lymphocytes another cell types. In this study, we investigated the molecular mechanisms that are involved in the apoptosis induced by TG in T cell hybridomas. Exposure to TG results in rapid induction of the orphan steroid receptor, Nur77, accompanied by apoptosis of T cell hybridomas. The expression of Nur77 in response to TG treatment is sensitive to cyclosporin A, implicating that activation of ***calcineurin*** is necessary for Nur77 expression. The TG-induced Nur77 expression is also inhibited by overexpression of Cabin1, an ***endogenous*** ***inhibitor*** of ***calcineurin*** and a corepressor of the transcription factor MEF2, suggesting that MEF2 activation is required for Nur77 expression. These results suggest that induction of Nur77 expression and apoptosis by TG are mediated by the same signaling pathways that are involved in T cell receptor-mediated thymocyte apoptosis, including the ***calcineurin*** pathway and Cabin1-MEF2 pathway.

L3 ANSWER 4 OF 6 MEDLINE DUPLICATE 4
ACCESSION NUMBER: 2001382225 MEDLINE
DOCUMENT NUMBER: 21152920 PubMed ID: 11231093
TITLE: Dscr1, a novel ***endogenous*** ***inhibitor*** of ***calcineurin*** signaling, is expressed in the primitive ventricle of the heart and during neurogenesis.
AUTHOR: Casas C; Martinez S; Pritchard M A; Fuentes J J; Nadal M; Guimera J; Arbones M; Florez J; Soriano E; Estivill X; Alcantara S
CORPORATE SOURCE: Down Syndrome Research Group, Medical and Molecular Genetics Center - IRO, Avia. de Castelldefels, km. 2.7, L'Hospitalet de Llobregat, 08907, Barcelona, Spain.
SOURCE: MECHANISMS OF DEVELOPMENT, (2001 Mar) 101 (1-2) 289-92.
Journal code: 9101218. ISSN: 0925-4773.
PUB. COUNTRY: Ireland
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200107
ENTRY DATE: Entered STN: 20010709
Last Updated on STN: 20010709
Entered Medline: 20010705

AB We have demonstrated that DSCR1 acts as a negative regulator of calcineurin-mediated signaling and that its transcript is overexpressed in the Down syndrome (DS) fetal brain. To evaluate the possible involvement of DSCR1 in DS, we have cloned the mouse gene and analyzed its expression pattern in the central nervous system (CNS). Early expression of Dscr1 is detected mainly in the heart tube and in the CNS in rhombomere 4 and the pretectum. From embryonic day 14.5 onwards, Dscr1 is widely distributed in the CNS but becomes more restricted as the brain matures. We confirmed its neuronal expression pattern in the adult, preferentially in Purkinje and pyramidal cells, by double labeling with glial fibrillary acidic protein. We also show that although Dscr1 is present in trisomy in the Ts65Dn

L3 ANSWER 5 OF 6 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: [redacted]

(Cain) to rat chromosome band 20p12 by fluorescence in situ hybridization
AUTHOR(S): Kim, H.; Jung, Y. K.; Jo, D. G.; Park, S. H.
CORPORATE SOURCE: Institute of Human Genetics, Department of Anatomy, Korea University College of Medicine, Seoul, 136-705, S. Korea
SOURCE: Cytogenetics and Cell Genetics (2000), 89(3-4), 236-237
CODEN: CGCGBR; ISSN: 0301-0171
PUBLISHER: S. Karger AG
DOCUMENT TYPE: Journal
LANGUAGE: English
AB ***Calcineurin*** inhibitor is a most potent ***endogenous*** ***inhibitor*** of ***calcineurin*** , and in its physiol. role is suspected to provide a docking site for ***calcineurin*** in its inactive form. Here we report the mapping of rat gene Cain to chromosome band 20p12 using fluorescence in situ hybridization.
REFERENCE COUNT: 10 THERE ARE 10 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L3 ANSWER 6 OF 6 MEDLINE DUPLICATE 5
ACCESSION NUMBER: 2000062818 MEDLINE
DOCUMENT NUMBER: 20062818 PubMed ID: 10593895
TITLE: Inhibition of calcineurin phosphatase activity by a calcineurin B homologous protein.
AUTHOR: Lin X; Sikkink R A; Rusnak F; Barber D L
CORPORATE SOURCE: Department of Stomatology, University of California, San Francisco, California 94143, USA.
CONTRACT NUMBER: DK40259 (NIDDK)
GM 47413 (NIGMS)
GM46865 (NIGMS)
SOURCE: JOURNAL OF BIOLOGICAL CHEMISTRY, (1999 Dec 17) 274 (51) 36125-31.
Journal code: 2985121R. ISSN: 0021-9258.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200001
ENTRY DATE: Entered STN: 20000204
Last Updated on STN: 20000204
Entered Medline: 20000127
AB ***Calcineurin*** , a Ca(2+)/calmodulin-stimulated protein phosphatase, plays a key role in T-cell activation by regulating the activity of NFAT (nuclear factor of activated T cells), a family of transcription factors required for the synthesis of several cytokine genes. ***Calcineurin*** is the target of the immunosuppressive drugs cyclosporin A and FK506 complexed with their cytoplasmic receptors cyclophilin and FKBP12, respectively. In this study we report that ***calcineurin*** is also the target of a recently identified Ca(2+)-binding protein, CHP (for ***calcineurin*** homologous protein), which shares a high degree of homology with the regulatory B subunit of ***calcineurin*** and with calmodulin. In Jurkat and HeLa cells, overexpression of CHP specifically impaired the nuclear translocation and transcriptional activity of NFAT but had no effect on AP-1 transcriptional activity and only a small (<25%) inhibitory effect on the transcriptional activity of NFkappaB. Further study indicated that CHP inhibits ***calcineurin*** activity. In cells overexpressing CHP, the phosphatase activity of immunoprecipitated ***calcineurin*** was inhibited by approximately 50%; and in a reconstituted assay, the activity of purified ***calcineurin*** was inhibited up to 97% by the addition of purified recombinant CHP in a dose dependent manner. Moreover, prolonged activation of Jurkat cells was

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     FILE 'MEDLINE, CAPLUS, BIOSIS, EMBASE, SCISEARCH, AGRICOLA' ENTERED AT
     17:36:27 ON 18 OCT 2002
L1         13784 S CALCINEURIN
L2            26 S L1 (P) (ENDOGENOUS INHIBITOR)
L3             6 DUPLICATE REMOVE L2 (20 DUPLICATES REMOVED)

=> s calcipressin
L4            18 CALCIPRESSIN

=> s csp1
L5           144 CSP1

=> s l5 (p) l1
L6             0 L5 (P) L1

=> s l4 (p) l1
L7            16 L4 (P) L1

=> duplicate remove l7
DUPLICATE PREFERENCE IS 'MEDLINE, CAPLUS, BIOSIS, EMBASE, SCISEARCH'
KEEP DUPLICATES FROM MORE THAN ONE FILE?  Y/(N):n
PROCESSING COMPLETED FOR L7
L8             4 DUPLICATE REMOVE L7 (12 DUPLICATES REMOVED)

=> s l8 not l3
L9             4 L8 NOT L3

=> d l9 1-4 ibib abs

L9   ANSWER 1 OF 4     MEDLINE
ACCESSION NUMBER:    2002300827     MEDLINE
DOCUMENT NUMBER:     22035335   PubMed ID: 12039863
TITLE:               The DSCR1 (Adapt78) isoform 1 protein   ***calcipressin***
                     1 inhibits   ***calcineurin***   and protects against acute
                     calcium-mediated stress damage, including transient
                     oxidative stress.
AUTHOR:              Ermak Gennady; Harris Cathryn D; Davies Kelvin J A
CORPORATE SOURCE:    Ethel Percy Andrus Gerontology Center, and Division of
                     Molecular and Computational Biology, University of Southern
                     California, Los Angeles, California 90089-0191, USA.
CONTRACT NUMBER:     AG16256 (NIA)
SOURCE:              FASEB JOURNAL, (2002 Jun) 16 (8) 814-24.
                     Journal code: 8804484. ISSN: 1530-6860.
PUB. COUNTRY:        United States
DOCUMENT TYPE:       Journal; Article; (JOURNAL ARTICLE)
LANGUAGE:            English
FILE SEGMENT:        Priority Journals
ENTRY MONTH:         200206
ENTRY DATE:          Entered STN: 20020604
                     Last Updated on STN: 20020611
                     Entered Medline: 20020607

AB   Although DSCR1 (Adapt78) has been associated with successful adaptation to
     oxidative stress and calcium stress and with devastating diseases such as
     Alzheimer's and Down syndrome, no rationale for these apparently
     contradictory findings has been tested. In fact, DSCR1 (Adapt78) has not
     yet been proved to provide protection against acute oxidative stress or
     calcium stress. We have addressed this question using cross-adaptation to
     H2O2 and the calcium ionophore A23187, stable DSCR1 (Adapt78) transfection
     and overexpression in hamster HA-1 cells, 'tet-off' regulated DSCR1
     (Adapt78) isoform 1 transgene expression in human PC-12 cells, and DSCR1
     (Adapt78) antisense oligonucleotides to test the ability of the DSCR1
     (Adapt78) protein product   ***calcipressin***   1 (a   ***calcineurin***
     [illegible] Adapt78 gene product   ***calcipressin***   1 to provide short term
     protection against acute oxidative stress and other calcium mediated
     stresses, whereas chronic overexpression may be associated with Alzheimer's
     [illegible]
```

L9 ANSWER 2 OF 4 MEDLINE
ACCESSION NUMBER: 2000436191 MEDLINE
DOCUMENT NUMBER: 20359261 PubMed ID: 10899116
TITLE: Identification and characterization of a highly conserved ***calcineurin*** binding protein, CBP1/ ***calcipressin*** , in Cryptococcus neoformans.
AUTHOR: Gorlach J; Fox D S; Cutler N S; Cox G M; Perfect J R; Heitman J
CORPORATE SOURCE: Departments of Genetics, Medicine, Microbiology, Pharmacology and Cancer Biology, and The Howard Hughes Medical Institute, Duke University Medical Center, Durham, NC 27710, USA.
CONTRACT NUMBER: 5T32AI07392 (NIAID)
RO1AI39115 (NIAID)
RO1AI42159 (NIAID)
+
SOURCE: EMBO JOURNAL, (2000 Jul 17) 19 (14) 3618-29.
Journal code: 8208664. ISSN: 0261-4189.
PUB. COUNTRY: ENGLAND: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200009
ENTRY DATE: Entered STN: 20000928
Last Updated on STN: 20000928
Entered Medline: 20000918

AB Calcineurin is the conserved target of the immunosuppressants cyclosporin A and FK506. Using the yeast two-hybrid system, we identified a novel calcineurin binding protein, CBP1, from the pathogenic fungus Cryptococcus neoformans. We show that CBP1 binds to calcineurin in vitro and in vivo, and FKBP12-FK506 inhibits CBP1 binding to calcineurin. Cryptococcus neoformans cbp1 mutant strains exhibit modest defects in growth under stress conditions and virulence, similar to but less severe than the phenotypes of calcineurin mutants. Saccharomyces cerevisiae mutants lacking the CBP1 homolog RCN1 are, like calcineurin mutants, sensitive to lithium cation stress. CBP1 shares a central peptide sequence motif, SPPxSPP, with related proteins in S.CEREVISIAE:, Schizosaccharomyces pombe, Drosophila melanogaster, Caenorhabditis elegans and humans, and peptides containing this motif altered calcineurin activity in vitro. Interestingly, the human CBP1 homolog DSCR1 is encoded by the Down's syndrome candidate region interval on chromosome 21, is highly expressed in the heart and central nervous system, and may play a role in calcineurin functions in heart development, neurite extension and memory.

L9 ANSWER 3 OF 4 MEDLINE
ACCESSION NUMBER: 2000386788 MEDLINE
DOCUMENT NUMBER: 20347037 PubMed ID: 10887154
TITLE: A conserved family of calcineurin regulators.
AUTHOR: Kingsbury T J; Cunningham K W
CORPORATE SOURCE: Department of Biology, Johns Hopkins University, Baltimore, MD 21218, USA.
CONTRACT NUMBER: GM53082 (NIGMS)
SOURCE: GENES AND DEVELOPMENT, (2000 Jul 1) 14 (13) 1595-604.
Journal code: 8711660. ISSN: 0890-9369.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200008
ENTRY DATE: Entered STN: 20000818
Last Updated on STN: 20000818
Entered Medline: 20000804

... the yeast protein Rcn1p ... the human homologs DSCR1 ... DAP-4 ... two independent functions of ***calcineurin*** in yeast: The activation of the transcription factor Tcn1p and the inhibition of the ... Ca^{2+} exchanger Vcx1p. Purified recombinant Rcn1p and DSCR1 bound ...

induced Rcn1p expression, suggesting that Rcn1p operates as an endogenous feedback inhibitor of ***calcineurin*** . Surprisingly, rcn1 null mutants exhibited phenotypes similar to those of Rcn1p-overexpressing cells. This effect may be due to lower expression of ***calcineurin*** in rcn1 mutants during signaling conditions. Thus, Rcn1p levels may fine-tune ***calcineurin*** signaling in yeast. The structural and functional conservation between Rcn1p and DSCR1 suggests that the mammalian Rcn1p-related proteins, termed ***calcipressins*** , will modulate ***calcineurin*** signaling in humans and potentially contribute to disorders such as Down Syndrome.

L9 ANSWER 4 OF 4 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 2002:642908 CAPLUS
TITLE: Regulation of ***calcineurin*** signaling in saccharomyces cerevisiae by sphingosine-1-phosphate and ***calcipressins***
AUTHOR(S): Birchwood, Christine Jo-Anne
CORPORATE SOURCE: Johns Hopkins Univ., Baltimore, MD, USA
SOURCE: (2002) 165 pp. Avail.: UMI, Order No. DA3028234
From: Diss. Abstr. Int., B 2002, 62(10), 4311
DOCUMENT TYPE: Dissertation
LANGUAGE: English
AB Unavailable

=> d his

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FILE 'MEDLINE, CAPLUS, BIOSIS, EMBASE, SCISEARCH, AGRICOLA' ENTERED AT 17:36:27 ON 18 OCT 2002

L1	13784	S CALCINEURIN
L2	26	S L1 (P) (ENDOGENOUS INHIBITOR)
L3	6	DUPLICATE REMOVE L2 (20 DUPLICATES REMOVED)
L4	18	S CALCIPRESSIN
L5	144	S CSP1
L6	0	S L5 (P) L1
L7	16	S L4 (P) L1
L8	4	DUPLICATE REMOVE L7 (12 DUPLICATES REMOVED)
L9	4	S L8 NOT L3

=> log y

COST IN U.S. DOLLARS	SINCE FILE ENTRY	TOTAL SESSION
FULL ESTIMATED COST	17.65	17.86

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)	SINCE FILE ENTRY	TOTAL SESSION
CA SUBSCRIBER PRICE	-0.62	-0.62

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